

AMENDMENTS TO THE CLAIMS:

1. (Currently Amended) A method for correlating the ability of a cell to bind IgA and cellular susceptibility to a disease, said method comprising:
identifying a Fc α RI genotype of said cell;
quantifying IgA binding by said cell expressing said Fc α RI genotype; and
comparing IgA binding by said cell and IgA binding by a second cell, said second cell expressing a second Fc α RI genotype, wherein correlation of the ability of said cell to bind IgA and cellular susceptibility to disease is indicated by a difference in IgA binding detected by said comparing.
2. (Original) The method of claim 1 wherein said Fc α RI genotype differs from said second Fc α RI genotype by a point mutation.
3. (Original) The method of claim 2 wherein said point mutation is a silent mutation.
4. (Original) The method of claim 2 wherein said point mutation is a frame shift mutation.
5. (Original) The method of claim 2 wherein said point mutation is a missense mutation.
6. (Original) The method of claim 3 wherein said silent mutation is in codon 87 of said Fc α RI genotype.

7. (Original) The method of claim 3 wherein said silent mutation is in codon 92 of said Fc α RI genotype.

8. (Original) The method of claim 5 wherein said missense mutation is at codon 132 of said Fc α RI genotype.

9. (Original) The method of claim 5 wherein said missense mutation is at codon 245 of said Fc α RI genotype.

10. (Original) The method of claim 5 wherein said missense mutation is at codon 248 of said Fc α RI genotype.

11. (Original) The method of claim 1 wherein said disease is selected from the group consisting of: periodontal disease, cancer, viral infection, bacterial infection, systemic lupus erythematosus, systemic vasculitis, IgA nephropathy, rheumatoid arthritis, systemic sclerosis, dermatomyositis, Hashimoto's thyroiditis, inflammatory bowel disease and Sjogren's syndrome.

12. (Original) The method of claim 1 wherein said cell is selected from the group consisting of: a neutrophil, a monocyte, a myeloid cell, and a mucus secreting cell.

13. (Original) A method for determining Fc α RI alleles specific to an individual human, said method comprising: genotyping DNA encoding Fc α RI for a polymorphism, said DNA being obtained from said individual human.

14. (Original) The method of claim 13 wherein said polymorphism affects IgA binding by a Fc α RI receptor.

15. (Original) The method of claim 13 wherein said polymorphism affects signal transduction.

16. (Original) The method of claim 13 wherein said polymorphism is a single nucleotide polymorphism.

17. (Original) The method of claim 13 wherein said polymorphism is a microsatellite polymorphism.

18. (Original) The method of claim 13 wherein said polymorphism is a splice isoform.

19. (Original) The method of claim 13 wherein said polymorphism is in the glycosylation sites of Fc α RI.

20. (Original) The method of claim 13 wherein genotyping utilizes PCR typing with a sequence specific primer for a polymorphic exon.

21. (Original) The method of claim 20 wherein said primer is selected from the group consisting of those shown in Example 4.

Claims 22-25 (Withdrawn)

26. (Currently Amended) A method of prognosticating a human immunoresponse to a disease, said method comprising:

establishing a correlation between a Fc α RI genotype and clinical outcome of said disease;

genotyping a patient for Fc α RI to yield a patient Fc α RI genotype;

comparing said Fc α RI genotype with said patient genotype; and

determining clinical outcome for said patient based on said patient genotype, wherein determining said clinical outcome is indicative of a human immunoresponse to a disease.

27. (Original) The method of claim 26 wherein genotyping utilizes PCR typing with a sequence specific primer for a polymorphic exon.

28. (Original) The method of claim 27 wherein said primer is selected from the group consisting of those shown in SEQ ID Nos. 1, 2, 3 and 4.

29. (Original) The method of claim 26 wherein genotyping comprises purifying Fc α RI expressing cells from said patient; extracting nucleic acids from said cells; and

determining whether the nucleic acid encodes a predetermined polymorphic FcαRI nucleic acid sequence.

30. (Original) The method of claim 29 wherein the nucleic acid is selected from the group consisting of: RNA and DNA.

Claims 31-33 (Cancelled)

34. (Original) A commercial package comprising reagents for identifying single nucleotide polymorphisms in a FcαRI genotype or phenotype together with instructions for use thereof as a test to identify individual susceptibility to a disease.

Claim 35 (Cancelled)

36. (New) A method for correlating response of a cell to binding FcαRI ligand and cellular susceptibility to a disease, said method comprising:

identifying a FcαRI genotype of the cell;

quantifying response to binding FcαRI ligand by the cell expressing the FcαRI genotype;

comparing response to binding FcαRI ligand by the cell and response to binding FcαRI ligand by a second cell, the second cell expressing a second FcαRI genotype, wherein correlation of the response of a cell to binding FcαRI ligand and cellular susceptibility to disease is indicated by a difference in response detected by the comparing.

37. (New) The method of claim 36 wherein the Fc α RI genotype differs from the second Fc α RI genotype by a point mutation.

38. (New) The method of claim 37 wherein the point mutation is a frame shift mutation.

39. (New) The method of claim 37 wherein the point mutation is a missense mutation.

40. (New) The method of claim 37 wherein the point mutation alters a phosphorylation site of Fc α RI.

41. (New) The method of claim 40 wherein the phosphorylation site is a casein kinase I phosphorylation site.

42. (New) The method of claim 36 wherein the response is activation of an enzyme.

43. (New) The method of claim 36 wherein the response is induction of phagocytosis.

44. (New) The method of claim 36 wherein the response is induction of oxidative burst.

45. (New) The method of claim 36 wherein the response is induction of cytokine production.

46. (New) The method of claim 36 wherein the response is release of collagenase.